

Dermatoses Due to Physical agents

- Ultraviolet light
- photodermatoses
 - ↳ polymorphous light eruption
 - ↳ Actinic prurigo
 - ↳ Chronic actinic dermatitis
 - ↳ Actinic Reticuloid
 - ↳ Solar Urticaria
 - ↳ Solar elastosis
 - ↳ Nodular elastosis & Cysts and Comedones

- photosensitivity
- phytophotodermatitis
- photoprotection
 - ↳ Sun Screens
- Diseases aggravated by Cold.
 - ↳ Frost Bite
 - ↳ Pernio Chilblains
 - ↳ Radiodermatitis
 - ↳ Erythema ab igne
 - ↳ Immersion Trench Foot

Amylee

1 Ultra violet Light

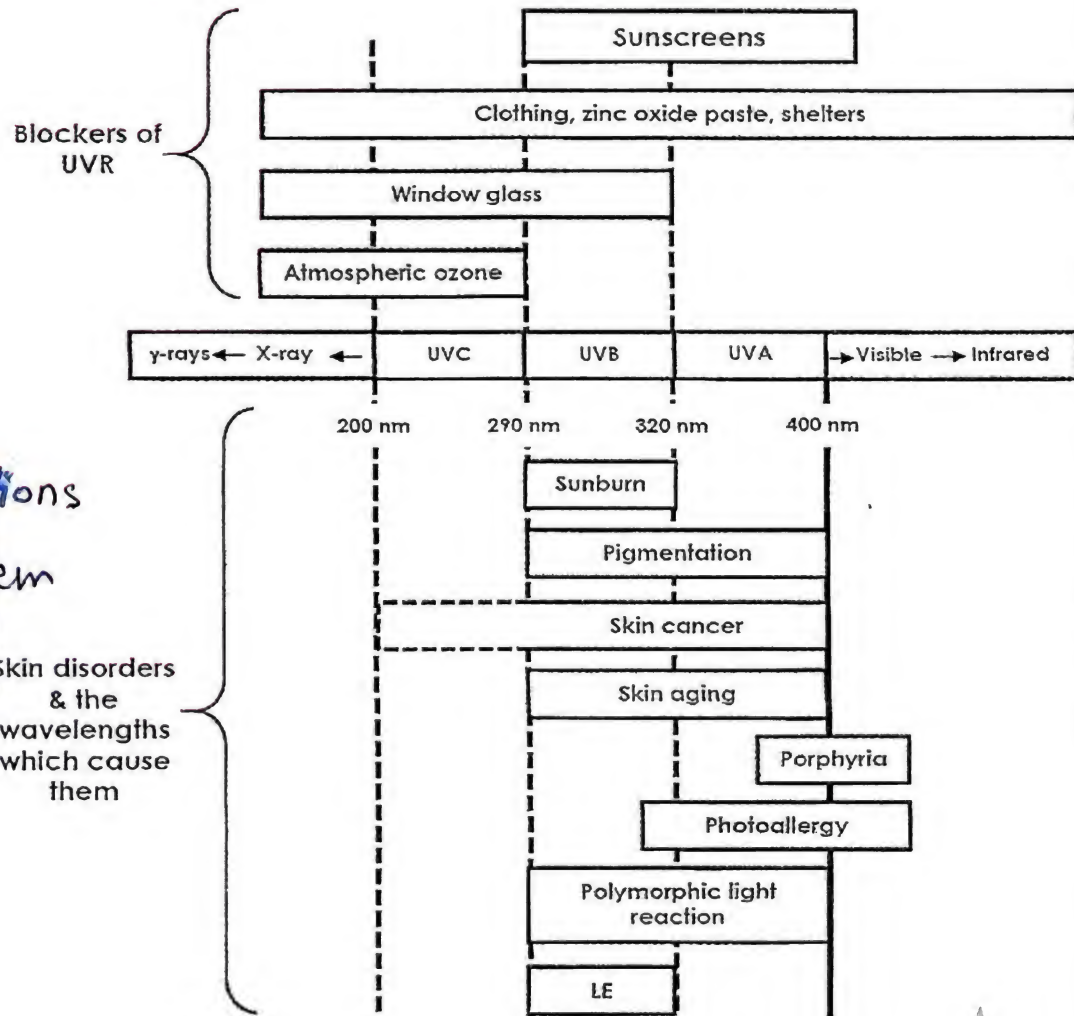
- Exposure of the skin to UV light has:
 - Acute short term effect
 - Chronic long term effect
 (Both - wavelength Dependant)
 - profound effect & non-erythrogenic Doses of UV light

- UV light induce several Different Types of DNA Damage
 - as Pyrimidine Dimers
 - oxidative guanine Base modifications

- UV light affect :- the skin's immune system exerting Both
 - pro
 - anti-inflammatory Response

- Several DNA Repair pathways are involved in the processing of UV-light-induced DNA Damage.

- nucleotide excision Repair
- Base excision Repair
- translesional DNA Synthesis
- Recombination Repair



- The solar spectrum at sea level includes :-
 - wavelength of about 290-3000 nm
 - The Ozone in the upper atmosphere provides :-
 - Barrier to wave lengths shorter than 290 nm
- ↓
- So the Earth's surface → Shielded from the shorter wavelengths which are → inimical to most forms of life
- The sunlight which reaches ground level :-
 - Consist of :-

- UV Radiation from 290 to 400 nm
- visible radiation from 400 to 700 nm

• Sun's UV Radiation:

- UVA 79.5%
- All of the UVC and much of UVB → absorbed by the oxygen and ozone.

UVR	UVC	= 200-290 nm - germicidal reaction. They do not reach the earth's surface.
	UVB	= 290-320 nm (erythemalogenic radiation). Some authors use 315 nm, rather than 320 nm, as the demarcation between UVA and UVB.
	UVA	= 320-400 nm (UVA1 "340-400 nm" and UVA2 "320-340 nm").
Visible light		= 400-760 nm.
Near infrared		760-1,000 nm
Far infrared		1,000-100,000 nm
Microwaves & radiowaves		>10 ⁶

• Normal Response of human Skin to Solar irradiation :-

★ Acute Reactions

1 Erythema :- (sunburn)

- Caused By → UVB
- after sun exposure → onset of Erythema occur within → 2 to 6 hrs
- max intensity occur → 15 to 24 hr after exposure
- Minimum Erythema Dose (MED) = is the minimal amount of energy required to produce a uniform, clearly defined erythema response usually at 24 hrs.
- The ability of UV to induce sunburn rapidly declines \propto increasing of wavelength

→ UVA can cause Erythema But The Dose Required is much Greater.

2 Pigmentation :-

Immediate

- By UVA and visible light
- V. soon after exposure
- lasts only few hrs
- D.t photo-oxidation of previously formed melanin

Delayed

- By UVB
- D.t formation of new melanin
- occur few days after exposure
- It protect skin from subsequent exposure

3 ↑↑ Thickness of Epidermis

- Occur few days after UVB or UVC exposure
- It protect against subsequent exposure

4 Immunologic alteration

- Cellular and molecular events in UV irradiation skin may be :-

3

• mediating Pro-inflammatory Immune Stimulating effect

- release of proinflammatory mediators By resident & non-resident skin cells e.g.
 - Serotonin
 - Prostaglandin
 - IL-1, IL-6, IL-8
 - TNF α

- Induction of antimicrobial peptides

(hypothesized to explain why UV-irradiation skin is Not prone to Bacterial infection)

5 Excision repair

UVR → Cause Damage of DNA → Followed By Excision repair which is a mechanism mediated By: endonuclease enzyme

Action spectra for photodamage

Effect	Spectrum of light		
	UVB	UVA	Visible
Sunburn	++++	+	
Photoaging	++++	++	?
Squamous cell carcinoma	++++	+	
Basal cell carcinoma	+++	?	
Cutaneous melanoma	++	+	
Photoimmune suppression	++++	++	
Photosensitivity	+	+++	+

• mediating Anti-inflammatory Immuno suppressive effect

- Depletion of langerhans cells or modulation of their antigen-presenting function
- Release of anti-inflammatory mediators By resident and non-resident skin cells e.g.
 - IL-10
 - α -MSH
- Induction of regulatory T-Cells (antigen-specific)

★ Chronic Reactions ★ [Chronic actinic Damage]

- Occur in: Blue eyes - Fair complexioned persons (Sailor's sign - Farmer's Skin)

• Photoaging (premature aging)

- changes in appearance and function of the skin
- as a Result of → repeated exposure rather than passage of time alone

- e.g. Coarseness, mottled pigmentation, solar elastosis, Telangiectasia, purpura, fibrotic depigmented areas (Pseudoscars)

- The only other environmental factor that produce premature aging of skin is: **Cigarette Smoking**

- Treatment: 0.1% tretinoin Cream

[4]

• Photo Carcinogenesis

(pre-malignancy - malignancy)

- e.g. Solar Keratosis
 - Bowen's Disease
 - Basal cell epithelioma
 - Sq. Cell Carcinoma
 - melanoma
 - actinic cheilitis
- disseminated superficial actinic porokeratosis

• Photo Carcinogenesis Cascade

UV → induced DNA Damage generates → mutations → accumulation of a sufficient number of mutation in critical genes → malignant transformation of single keratinocyte or melanocyte formation of skin cancer

• Cutaneous signs of Significant Photodamage

1. Solar elastosis
2. Nodular elastosis
3. weathering nodules
4. Cutis rhomboidalis nuchae
5. Colloid milium
6. Cutan. melanoma (lentigo maligna)
7. Solar lentigines: ephelides
8. Squamous Cell Carcinoma
9. Solar purpura - hemosiderin deposition
10. Acquired elastotic hamangioma
11. Actinic Keratosis
12. Actinic Comedonal plaque
13. Basal cell carcinoma
14. Pseudoscars
15. Poikiloderma of Civatte
16. Grover's Disease

2 Photodermatosis

• Skin Disorders Induced or exaggerated By light .

Classification of photodermatoses

Immunologically mediated photodermatoses	Drug- & chemical-induced photosensitivity
<ul style="list-style-type: none"> Polymorphous light eruption. Chronic actinic dermatitis. Actinic prurigo. Hydroa vacciniforme. Solar urticaria. 	<p>Exogenous:</p> <ul style="list-style-type: none"> Phototoxicity: Systemic & topical. Photoallergy: Systemic & topical. <p>Endogenous:</p> <ul style="list-style-type: none"> Cutaneous porphyria.
Hereditary photodermatoses	Photoaggravated dermatoses
<p>Caused by defects in nucleotide excision repair:</p> <ul style="list-style-type: none"> Xeroderma pigmentosum. Cockayne syndrome, including cerebro-oculo-facio-skeletal syndrome. Trichothiodystrophy. Ultraviolet light sensitive syndrome. <p>Caused by double strand break repair defects:</p> <ul style="list-style-type: none"> Rothmund-Thomson syndrome. Bloom syndrome. <p>Caused by abnormal chemical substances:</p> <ul style="list-style-type: none"> Smith-Lemli-Opitz syndrome. Hartnup disease. <p>Others:</p> <ul style="list-style-type: none"> Kindler syndrome. 	<ul style="list-style-type: none"> Atopic dermatitis. Darier-White disease. Dermatitis herpetiformis. Herpes simplex infection. Lupus erythematosus & neonatal lupus erythematosus. Juvenile dermatomyositis. Pellagra. Psoriasis.

• Laboratory Evaluation of photosensitivity :-

1. Routine histology
2. Anti-nuclear antibodies
3. Anti-RO/SSA La/SSB antibodies
4. plasma porphyrins followed By Complete porphyrin profile → if +ve
5. phototesting to UVA, UVB, visible light
6. photopatch testing
7. in infants and children → urinary amino acids

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• DD of photodermatoses most Commonly associated with different age groups :-

1- when the ptn is a Child :-

- Juvenile spring eruption
- childhood porphyrias
 - ↳ erythropoietic protoporphyria
 - ↳ congenital erythropoietic protoporphyria
- Actinic prurigo
- Hydroa vacciniforme
- Genodermatoses

2 - when the ptn is an adult -

- polymorphous light eruption
- Drug-induced photosensitivity
- Solar Urticaria
- lupus erythematosus
- Porphyria Cutanea Tarda

3- when the ptn is old -

- Chronic actinic Dermatitis
- Drug induced photosensitivity
- Dermatomyositis

④ Phototesting :

- Confirm The presence of a photosensitivity Disorder
- most helpful for the Diagnosis of Immunologically-mediated photodermatosis
- using : opaque template with several windows
 - the uninvolved skin of the Back or abdomen \rightarrow exposed to varying doses of UVA, UVB or visible monochromatic or Broad-spectrum radiation.
- Following light exposure \rightarrow the first reading : performed in 20 minutes to \rightarrow Detect urticarial lesions as seen in Solar Urticaria

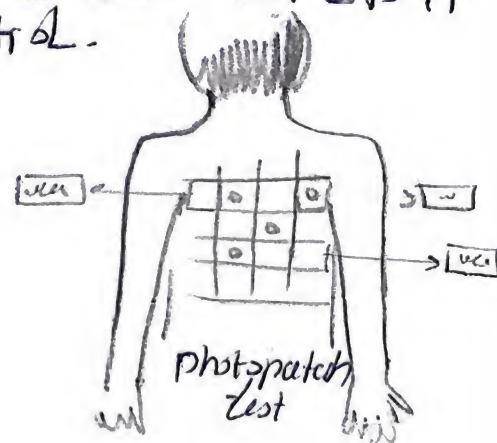
⑤ Photopatch Testing :

- evaluate pts w photoallergic Contact Dermatitis
- it's similar to standard patch test used for evaluation of allergic Contact Dermatitis
- Notable Difference include:

irradiation of the patch site w UVA in the photopatch testing

- Duplicate sets of photoallergen panels are placed on the Back and the sites are then covered w an opaque material \rightarrow to protect them from exposure to light.

- After 24 hr \rightarrow one of the panels is irradiated w a Dose of 10 J/m^2 of UVA OR 50% of MED-A. The other panel act as Control.



Expected phototest & photopatch test results*

Disorder	MED for UVA	MED for UVB	Visible light	Photopatch test
Polymorphous light eruption	NL / \downarrow	NL / \downarrow	NL	Negative
Chronic actinic dermatitis	\downarrow	\downarrow	NL / \downarrow	Negative / positive
Solar urticaria	Urticaria	Urticaria	Urticaria	Negative
Phototoxicity	\downarrow	NL	NL	Negative
Photoallergy	\downarrow	NL	NL	Positive

NL: Normal

1 Polymorphous light Eruption: PMLE

- most common type of photodermatosis
- abnormal reaction to sunlight (UVB-UVA - rarely visible light)
- It affects: young adults, light complexion from May to October every year.

• Clinically:

- 1- eruption appear 4 hrs - 4 days after sun exposure in the exposed areas:- Face - V area of chest, neck, arm

2. 4 Types present:

- papular - papulovesicular - plaque -
Diffuse Erythematous
 - 3- No Scarring or atrophy
- Sally indurated Plaque → DLE

• Juvenile Spring eruption

- The helixes of the ears (Boys) - Their ears more exposed.
- with vesicles
- the tendency for eruption ↓↓ as the summer or sunny vacation proceeds "Hardening"

- Histopathology: all types are non-specific except plaque type:

patchy lymphocytic infiltrate → resembling DLE
But the patchy infiltrate is perivascular
absent basal hydropic Degeneration.

• Diagnosis:

- +ve phototesting: wave length $< 320 \text{ nm}$
- ve lupus Band test → more important

• Prophylaxis: = Induction of Hardening

1. Initiated During spring
2. 2-3 sessions weekly → of NB-UVB, PUVA about 5 weeks

+

Oral prednisone (1 mg/kg) During the initial 7-10 Days of the ttt → to minimize photoexacerbation

- 3- pt then asked to expose themselves to noonday sunlight for 15-20 minutes (without sunscreen) weekly for the remainder of the sunny season to → maintain the hardened state

• For NB-UVB: initial starting Dose = 50-70% the MED

The Dose then ↑↑ By 10-15% per Δ

• For PUVA: 0.5-0.6% mg/kg of 8-methoxy-psoralen → given 1 hr before UVA exposure
 The starting UVA Dose range 0.5-3 J/cm²
 ↓
 Depending on Skin photoType.

The UVA Dose is ↑↑ By 0.5-1.5 J/cm² per Δ

• Treatment:

• Topically: Steroids - Sunscreens

• Systemic: Antihistamines, Systemic Steroids
 antimalarials

2 Actinic Prurigo

Hutchinson's Summer prurigo

• Onset: appear During childhood - girls

• Lesion: prurigo-like papules, small vesicles, pitted scars → present throughout the year

• Worst in summer involving exposed and covered areas

• frequent personal - family history of atopy

• Management: photoprotection - PUVA
 NB-UVB - Calcineurin inhibitors

Actinic prurigo vs PMLE

Feature	Actinic prurigo	PMLE
Starts	2-9 years of age	9-29 years of age
Relation to UV exposure	Often noted later	Clear
Pruritus	Severe, persistent	Transient
Covered areas of body affected	Frequent	Rare
Scar formation		
Ears affected		Never
Distal one-third of nose affected		
Plaques on the philtrum		
Cheilitis of the lower lip		
Conjunctivitis	Possible	Never
HLA association	DR4/DRB1*0407	None
Prophylaxis	Difficult	Easy

3 Hydroa vacciniforme

• onset: Early childhood

• lesion: Deep seated umbilicated vesicles on exposed areas

• Healing: with large varioliform scars

• E. Barr viral infection → detected in some pts

• Management:

- Very careful photoprotection

- B. Carotene - antimalarials - azathioprine

Thalidomide - Cyclosporine

Dietary fish oil

8

4 Chronic Actinic Dermatitis CAD

- photosensitive eczema (PE), persistent light reactivity (PLR), chronic photosensitive dermatitis (CPD) → precursors of actinic reticuloid

• The Diagnosis of CAD depends on:

- 3
- ↳ 1-persistent eczematous eruption of sun-exposed skin with possible extension into non-exposed area
 - ↳ 2-photosensitivity to UVB and often also longer wave length
 - ↳ 3-Histologically → evidence of chronic eczema, with or without eczema-like changes

• Pathogenesis:

- Transition from photoallergy to persistent light reactivity
- alteration ~~from~~ of some normal skin components during photoallergic reactions

Become → antigenic on its own.

- If CAD supervenes → UVB irradiation may Trigger the Delayed-type hypersensitivity Response at any site By Formation of an antigenic photoproduct from the endogenous carrier protein alone or in the absence of exogenous initiating agents.

- +ve patch or photopatch test → common

• Management:

- By: photoprotection
 - avoidance of (photo) contact sensitizer
 - intermittent oral and Topical corticosteroids
 - Topical tacrolimus
 - Low-Dose PUVA
 - Cyclosporine
 - azathioprine
 - Mycophenolate Mofetil
 - Probability of spontaneous resolution is 10% over 5 yrs
- 9 20% over 10 yrs

5] Actinic Reticuloid

• lesion: severe persistent photosensitivity with: Erythema, edema, Striking "Leonine" thickening of the light-exposed skin of the face, neck, hands

• Prior photosensitive atopic Dermatitis → more to develop actinic Reticuloid

• Occur: almost exclusive in elderly men

• There are lichenified plaques first in the exposed area

Later: The eruption gradually spreads to cover most of the skin surface

↓
Erythroderma + generalized LN enlarge

• There is Thickening (deep furrows) and Hyperpigmentation of the exposed areas

• Itching → severe

• The action spectrum → is UVA

Histopathology:

- Band like infiltrate of lymphoid and histiocytes in upper Dermis → extend into Lower dermis or invade into epidermis → aggregates → resembling "pautrier microabscesses"

- Benign - reversible → T-cell in skin lesions and circulation are the suppressor (OKT-8) Type →

- where in ETCL → its of helper (OKT-4) type

Treatment:

• systemic Steroids
• PUVA

• Azathioprine
• Sunscreens → ineffective

Leonine facies - associated dermatologic disease

• Scleromyxedema
• systemic amyloidosis
• lipid proteinosis
• Lepromatous leprosy
• Lishmaniasis
• Cut lymphoma

• Leukemia cutis
• Mastocytosis
• Sarcoidosis
• Actinic Reticuloid form of chronic actinic Dermatitis
• pachydermoperiostosis

6 Solar Urticaria:-

- physical urticaria → induced By :- Sun exposure
- lesions appear within 5-10 minutes and resolve over 1-2 hrs.
- Chick By : Immediate Urticarial Response to sunshine
- Occurring : in sun-exposed skin.
- Systemic symptoms : occur if there is sufficient mast cell release.
- The action spectrum generally → includes :- Both UVA, visible light rarely UVB.
- slight female predominance
- with onset at any age -
- Treatment : → plasmapheresis - IV Ig
- avoidance - sunscreens
- High Dose - non sedating antihistamines
- PUVA → cause depletion of mast cell contents

Classification of solar urticaria

	Mechanism	Action spectrum
Type I	Allergic	UVB
Type II	Unknown	UVA
Type III	Unknown	Visible light
Type IV	Allergic	Visible light
Type V	Unknown	UVA, UVB, visible light
Type VI	Protoporphyrin	Visible light

- Pathogenesis:

- Chromophore → present in the skin or in the circulation, OR Both may absorb radiation
- photoallergen formation
- IgE mediated Hypersensitivity to this photoallergen then develops with mast cell activation.

7 Solar (actinic) elastosis:-

- occur after prolonged exposure to Sun rays . Specially : fair-skinned persons.
- Small yellowish papules - plaques → Develop on Face and Back of Hands.
- The skin show : Deep furrows and wrinkles
- Subject to Development of actinic Keratoses and Carcinomas

• Cutis rhomboidealis nuchae:

- The skin on the Back of the neck → Become thickened and tanned with exaggerated normal skin markings

• Histopathology:

H & E

- upper dermis → Basophilic degeneration of the collagen
- Separated from the atrophic epidermis by narrow Band of Normal collagen

By elastic tissue stain

- aggregates of Thick, interwoven Bands of elastotic material which is newly formed by :- fibroblasts which are no longer Capable of producing normal elastic fibers or collagen

8 Nodular elastosis with Cysts, Comedones:-

[Favre-Racouchot Syndrome]

- some pts. e pronounced solar elastosis of the facial skin show: especially lateral to the eyes

multiple comedones - as well as → yellowish nodules, Contain: Central Comedo.

- Variant of nodular elastosis :- e Cysts and Comedones is the "actinic comedonal plaque"

its Found as: Solitary plaque on - sun damaged Skin of either the arms or the face

- The plaque show: small nodules - Dilated follicles

• Histopathology:

- pronounced solar elastosis
- Dilated pilosebaceous openings
- large - round - Cysts → lined By: flattened epidermis and Represent: greatly extended Hair follicles.

Photosensitivity

• number of substances known as photosensitizers → induce - abnormal reaction in skin exposed to sunlight

• These photosensitizers come in contact with the skin through → external or internal routes

I) Phototoxic reactions :-

• ch. ch By: exaggerated sunburn-like reaction.

• Caused By: Systemic agents.

• The Cutaneous porphyrias are an example of

↳ phototoxicity induced by endogenous agents.

• It is non-immunologic Reaction Can be elicited in the majority of individuals 2-6 hrs after exposure of the photosensitizers →

• The cytotoxic effect are due to generation of-

1- O_2 free Radicals - superoxide anions - hydroxyl radicals

2- stable photoproducts (e.g. chlorpromazine, tetracyclines)

3- photoadducts (e.g. psoralens)

4- Inflammatory mediators (e.g. porphyrins)

• Phototoxic Drug Reaction :-

Severe sunburn without itching e.g. :- sulfonamides, psoralen, Doxycycline

• Phototoxic Contact Dermatitis :-

• with coal tar derivatives and psoralens as: Oil of bergamot → which are present in some perfumes →

"Berloque or perfume Dermatitis" → manifested by streaking or drop-shaped lesions on sides of neck

II) Photoallergic reactions :-

• photallergy presents with :- eczematous lesions, related to topical photoallergens

- It's a cell mediated - Delayed immunologic response Can elicited only in small number of individuals who

have been sensitized By previous exposure to photosensitizer Drugs and at the same time to light as in allergic contact dermatitis

- the role of light consists in:- altering either the hapten itself or the avidity with which the hapten → Combines with the Carrier protein → to Form Complete photoantigen.

- photoallergic Drug eruption causes a: photocontact Dermatitis in all light-exposed areas.

- it- Cause → Itching

• photoallergic Drug reactions:

• e.g: Chlorothiazides • Tolbutamide
phenothiazines (chlorpromazines)

• photoallergic Contact Dermatitis
sulfathiazides - antihistamines

• Histopathology:-

↳ ① Phototoxicity: Scattered necrotic Keratinocytes 'sunburn cells' • Dermal

infiltrate of primarily < lymphocytes
neutrophils

↳ ② photoallergy:

- Epidermal spongiosis + Dermal lymphohistiocytic infiltrate

- Indistinguishable from other causes of spongiotic Dermatitis

• Treatment of photosensitivity :-

1- avoiding the offending agents → if not possible

2- Strict photoprotection (UVB-UVA) → required

3- for phototoxic reactions → analgesia helpful

4- Topical corticosteroids for severe flares →
short courses of systemic corticosteroids

↓
Can be used for photoallergic Dermatitis

5- evening dosing of phototoxic Drug →
Can be Done so that peak systemic levels occur During the night.

phyto-photo-dermatitis

• Definition: Form of phototoxicity Caused By:
The Combination of a Topical or Oral
photosensitizing agents.

[Furocoumarins "psoralens, angelicins" →
most common agents]

• Followed By: exposure to the appropriate
wavelength of UV radiation.

- Limes - Celery - rue → the most common
causes

- it's Not an immunologic Reaction →
So No prior sensitization is necessary
and anybody can be affected.

- photoallergic reaction → to a plant
is exceedingly rare.

• Clinical Features :-

① Cut-sensitivity to UV light peaks
30-120 minutes after contact with
furocoumarins

② Bizarre Configurations of Erythema, edema
and bullae → appear after 24 hrs & peak 72 hrs.

③ These painful, non-pruritic Reactions →
more often seen in mid to late summer
when psoralen concentrations are highest in the
offending plants, more skin is exposed to
Direct sunlight.

④ Hyperpigmentation appears 1-2 weeks later
and lasts months to years.

⑤ Low-Dose UVA or psoralens → Cause:-
Hyperpigmentation, without a preceding Vesicular
or erythematous eruption.

• Treatment :-

1- prevent Contact w/ offending plants

2- if Contact occurred → prompt washing with
Soap and water → may prevent a reaction

	<ul style="list-style-type: none"> • Solar urticaria • Erythropoietic protoporphyria
Papule	<ul style="list-style-type: none"> • Polymorphous light eruption • Actinic prurigo • Chronic actinic dermatitis
Vesicle	<ul style="list-style-type: none"> • Polymorphous light eruption • Juvenile spring eruption • Porphyria cutanea tarda • Variegate porphyria • Coproporphyria • Phototoxicity • Photoallergy • Hydroa vacciniforme
Erosion, crust	<ul style="list-style-type: none"> • Actinic prurigo • Hydroa vacciniforme • Porphyrias (PCT, VP, CEP, HC)
Eczema &/or lichenification	<ul style="list-style-type: none"> • Chronic actinic dermatitis
Erythema	<ul style="list-style-type: none"> • Phototoxicity
Scars	<ul style="list-style-type: none"> • Hydroa vacciniforme • PCT • VP • CEP

CEP: Congenital erythropoietic porphyria, HC: Hereditary coproporphyria,
PCT: Porphyria cutanea tarda, VP: Variegate porphyria.

& action spectra of photosensitive skin diseases

	Spectrum of light		
	UVB	UVA	Visible

Photoprotection : Sunscreens

Sun Screens:

• a preparation that attenuate UV wavelengths that would interact with molecules in the skin (chromophores) → to generate → photochemical reactions → photobiologic changes

Mechanism :-

- sunscreens forms a film or coat on the surface of the stratum corneum

absorb the photons reaching the skin

- The energy absorbed by the organic agents
↓
Converted to
↓
non-damaging energy and dissipated primarily as heat

scatter the photons reaching the skin

Scattering of Radiation is based on particle size

Combination of Both types

offering High levels of protection against Both UVB and longer wavelengths

Sunscreens Applications :-

1. applied 1/2 hr before sun exposure
2. The proper quantity of sunscreen to cover the skin of an adult is 30 mL or 2 tablespoons
3. strategies to improve compliance:-
 - applying the product twice → to gain the proper concentration → Reducing the occurrence of "Skip" areas
 - or use a product with twice the SPF desired

Active ingredients :- in sunscreens

1. physical:

- e.g.: titanium dioxide, Talc, Zn oxide
- which act as reflectors and scattering agents
- form an opaque barrier to all sunlight But They are cosmetically Unacceptable
- Can discolor clothes
- may promote miliaria-folliculitis
- limited protection when a person sunbaths for hrs

2-Chemicals:

- non opaque - colorless - contain absorbing chemical

e.g:-

(*) para-aminobenzoic acid PABA and PABA esters:-

- penetrate st. Corneum → where it can remain attached to proteins through hydrogen bonding.

- It can give protection even after Bathing, Swimming, perspiring

- PABA → Highly effective UVB absorbers

(*) Benzophenones: Oxybenzone

They absorb UVB and shorter wavelength

(*) Cinnamates: They absorb mostly UVB and some UVA

- They Don't Bind to st. Corneum
So more easily Removed

(*) Dibenzoylmethanes: more effective UVA screen
But poor UVB absorber

3-other agents:-

(*) Dihydroxyacetone: compound most commonly used in Sunless tanners

(*) Iron Oxide: offers protection across the UV spectrum and into the visible range, used as a colorant.

(*) Other active ingredients

- added to sunscreens → with claims of ↑ efficacy of the finished products e.g: Antioxidants, vit E, C, green tea

(*) Boosters:

They are Not UV filters, But they serve to ↑ the protective nature of sunscreen. Example:
● Tiny spheres: Scatter incoming radiation and agents that ↑ the spread of the sunscreen on the skin → greater protection

● Efficacy of Sunscreens:-

- Depends on: Vehicle, pH stability - SPF - Type of skin environment, sweating - Substantivity (The sunscreen Remain effective after prolonged sweating or swimming)

Determination of the sun protection factor (SPF)

- **Instrumentation:** Light source which mimics solar spectrum.
- **Procedure:** Determine minimal erythema dose (MED) in protected* & unprotected skin.
- $$SPF = \frac{MED \text{ protected}}{MED \text{ unprotected}}$$
- **To test substantivity** – after application & before MED testing:
 - Water resistant (40): 2 x 20 min water immersions (whirlpool bath)†.
 - Water resistant (80): 4 x 20 min water immersions (whirlpool bath)†.

• a product e⁻ SPF of 10 → would allow 10 times as much time in the Sun e⁻ The same Resultant level of Erythema as without The product in a given individual

• an SPF 20 similarly would allow 20 times as much exposure with The same Result But → this doesn't mean that the SPF-20 product absorbs twice as much Radiation as the SPF 10 products

☑ Sun protection is advised to:

- Fair skin (Type 1-2)
- Sun sensitivity disorders
- Personal or family History of skin cancer
- outdoor occupations

Labeling of sunscreens – ultraviolet B (UVB) and ultraviolet A (UVA) protection (2011)

Ultraviolet B (UVB) protection
• Sun protection factor (SPF): Up to 50+
Ultraviolet A (UVA) protection
• Either no label or broad spectrum.
• Broad spectrum can be used if critical wavelength is ≥ 370 nm.
Substantivity
• Water resistant (40 minutes) or water resistant (80 minutes).
• No use of the following designations: "Waterproof", "sunblock", "all day protection", or "sweat proof".
Additional labeling
• If a sunscreen has SPF ≥ 15 and is broad spectrum, then can state that the product can help to reduce the risk of skin cancer & the risk of early skin aging, when used regularly & as directed in combination with other sun protection measures.
• If the SPF of the sunscreen is < 15 or it is not broad spectrum, then it must have the following skin cancer/skin aging alert: "Spending time in the sun increases your risk of skin cancer & early skin aging. This product has been shown only to help prevent sunburn, not skin cancer or early skin aging".

❑ Adverse effects of Sunscreens:

- minor skin irritation → common
- Allergic contact Dermatitis → Rare
- Effect on vitamin D
 - Sunscreens that are effective at blocking UVB photons → may block some cut. vit D synthesis
 - a Diet that include vit D rich food and moderate amount of supplements combined w/ modest amount of everyday sun exposure → is enough to maintain adequate serum vit D level → even if the individual photoprotects w/ sunscreen.

II Systemic photoprotections

e.g:- Beta-carotenes
antimalarials

Diseases Caused or aggravated By Cold

① Reaction to extreme Cold

- Frost Bite: occur when the skin temperature drops below about -2°C
- Clp:
 - Erythema - edema - numbness → Replaced By Marked hyperemia & pain
 - occurring mainly on: Exposed parts (Ears - nose - fingers - toes)
 - Tissue Freezing, vasoconstriction and inflammatory mediators Release
- Pathophysiology →
- Treatment → Rapid Rewarming in warm water Bath is the Cornerstone

② Abnormal Reaction to Cold

- Chilblain (pernio)

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- Acrocyanosis: persistent Dusky discoloration of Hands & feet with coldness in young girls
- Erythrocyanosis
- Livedo - Reticularis: Cyanotic discoloration of skin & network pattern
- Raynaud's disease: also in LE, Scleroderma, Dermatomyositis - RA, Cryoglobulinemia
- Cold Urticaria
- Cryoglobulinemia
- Neonatal cold injury or sclerema
- Cold panniculitis

pernio chilblains

- D.f: abnormal inflammatory Response to cold, damp, non-freezing conditions
- Pathogenesis:
 - 1- Vascular Origin
 - 2- in children → associated w/ Cryoglobulins or Cold agglutinins

-Epidemiology:

- Exposure to cold & wet conditions specially in area where Homes lack central heating
- women & children & the elderly are most commonly affected
- Elderly pt may have prolonged course
- younger pt → improve spontaneously.

- Clinical Features:

- 1- Single or multiple Erythematous to Blue-violet macules, papules, nodules
- 2- In severe cases → Blistering - ulceration occur
- 3- Bilaterally Symmetrically on the distal Toes - fingers & less often on the heels, nose & ears
- 4- Deep pernio - affect thighs, calves and Buttocks & Blue-erythrocyanotic plaques
- 5- Symptoms: Itching & Burning or pain [22]
- 6- lesions often Resolve in 1-3 weeks except among elderly pt & venous insufficiency

-Pathology (non specific)

- Dermal edema + superficial - deep lymphohistiocytic infiltrate & peri-ecrine accentuation
- Necrotic Keratinocytes - lymphocytic vasculitis

-Laboratory evaluation:-

- 1- CBC → to exclude hemolytic anemia and myelomonocytic leukemia
- 2- Cryoglobulin, Cold agglutinin and cryofibrinogen levels to eliminate Cold-sensitive dysproteinemia
- 3- Serum protein electrophoresis and immunofixation electrophoresis to exclude a monoclonal gammopathy

-Treatment:

- 1- adequate clothing - avoidance of cold & damp conditions
- 2- Avoidance of smoking

3 - Nifedipine

4 - Nicotinamide, phenoxybenzamine
Sympathectomy, erythemogenic
UVB phototherapy

Radio dermatitis

- The effect of ionizing Radiation on the cells depends upon the amount and intensity of Radiation and the type of individual cell.

- These effects vary from arrest of mitosis, chromosomal damage, cell death

Early acute

- after large Doses of X-radiation or Radium
- Erythema develops within about week.
- heat & desquamation, pigmentation
- If dose High enough → Painful Blisters develop.
- Healing & atrophy - telangiectasia, irregular Hyperpig.
- very Large Dose → ulceration occur

Erythema ab igne

→ prolonged exposure to moderate Heat → emanating from fireplaces, radiators, heating pads

Causes → Persistent Reticular Erythema with or without pigmentation

→ The shins - Buttocks → most common site

→ possible Development of → Cutaneous Sq. Cell Carcinoma or Merkel Cell Carcinoma

late (chronic)

- Occur from few months to many years after the administration of fractional doses of X-rays or Radium
- Skin → atrophy, telangiectasia, irregular hypo- & hyperpigmentation
- ulceration - foci of Hyperkeratosis

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Immersion Foot

- Injury → occur after continuous exposure of Feet to moist, occluded conditions
 - Cold water & warm water - tropical variants exist
- The underlying pathological process → over-hydration of str. Corneum
- Neuropathy may persist indefinitely
- Feet exposed to immersion injury → are more sensitive to Re-injury
- Prevention is the Best !!!